ORGANOCHLORINE PESTICIDES IN THE ENVIRONMENT

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ABSTRACT

Each year for nearly 20 years, thousands of pounds of persistent organochlorine pesticides have been applied to outdoor areas in many countries. These compounds may last for a very long time in the environment, and be carried by wind, water, and animals to places far distant from where they are used. As a result, most living organisms now contain organochlorine residues. This paper constitutes a selective review of the literature concerning the occurrence, distribution, and effects of organochlorines in the environment.

Highest concentrations generally occur in carnivorous species. Thus predatory and fish-eating birds ordinarily have higher residues than do herbivores; quantities are similar in birds of similar habits in different countries.

Any segment of the ecosystem - marshland, pond, forest, or field - receives various amounts and kinds of pesticides at irregular intervals. The different animals absorb, detoxify, store, and excrete pesticides at different rates. Different degrees of magnification of pesticide residues by living organisms in an environment are the practical result of many interactions that are far more complex than implied by the statement of magnification up the food chain. These magnifications may be millions of times from water to mud or only a few times from food to first consumer.

Direct mortality of wild animals as an aftermath of recommended pesticide treatments has been recorded in the literature of numerous countries. However, accidents and carelessness also accompany pesticide use on a percentage basis and are a part of the problem. More subtle effects on the size and species composition of populations are more difficult to perceive in time to effect remedies. The possibility of ecological effects being mediated through changes in physiology and behavior has received some attention and has resulted in some disquieting findings. These include discovery of the activity of organochlorines in stimulating the breakdown of hormones or in acting directly as estrogens, their involvement in embryonic and early post-embryonic toxicity, interferences with antibody formation, effects on behavior, and interactions with stress such as nutritional deficiencies or food deprivation. Delayed mortality long after dosage ceased has shown the serious effects of storage of organochlorines in fat. DDT has been suggested as the indirect cause of a reduction of egg-shell thickness that occurred in the midforties in association with failing reproduction and population decline of certain predatory birds.

The impact of these new components of the environment has appeared as death, reproductive impairment, disruption of species balance, and behavioral alteration, but the overall effects on the environment have not been determined. Research should be aimed at interpretation of the significance of pesticide residues to survival and reproduction, to assessment of levels of pesticides in critical environments, and to the kinetics of pesticides in individuals and the ecosystem.

ORGANOCHLORINE PESTICIDES IN THE ENVIRONMENT

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Man's application of synthetic organochlorines to the environment on an economic scale started in the midforties, and thus began an intriguing ecological story, still unfolding after more than two decades. In this time the organochlorine pesticides have become part of the environment both living and nonliving, including the air, the rain, the Nation's rivers, and man himself (Abbott et al., 1966; Breidenbach, 1965; Hayes and Dale, 1963; Hunter et al., 1963; Quinby et al., 1965; Tabor, 1966; Weaver et al., 1965; Weibel et al., 1966; Wheatley and Hardman, 1965).

The purpose of this paper is to set forth some of the important things that are known about organochlorine pesticides as they relate to the natural world and some of the things that are not known. Along with new material this paper combines much of the material presented in two previous papers (Dustman and Stickel, 1966 and 1967). It is a selective review rather than a comprehensive one. The references cited are but a fraction of those available. They were selected to clarify the state of knowledge and to point up research needs relative to organochlorine residues in the natural environment. With a few exceptions, publications that appeared after February 1968 are not included.

USE OF ORGANOCHLORINES

Each year for nearly 20 years, thousands of pounds of persistent organochlorine pesticides have been applied to the environment in many countries. Worldwide occurrence of residues in man and nature is not surprising.

Annual statements of the U.S. Department of Agriculture (1953-1967) show the use of organochlorines in the United States (table 1). In 1958-59, 78 million pounds of DDT and 73 million pounds of chemicals of the aldrin group were used in the United States. By 1964-65, use of DDT had declined to 53 million pounds, but use of the more toxic chemicals of the aldrin group had increased to 80.5 million pounds. Organochlorines exported from the United States account for only a part of their use in other countries, yet 99 million pounds of DDT and 20 million pounds of chemicals in the aldrin group were exported in 1964-65. Thus, persistent chemicals are being added to the environment by the millions of pounds each year.

MOVEMENT OF ORGANOCHLORINES IN THE ENVIRONMENT

Organochlorine pesticides last a very long time in the environment, but not necessarily at the site of application. Wind, water, and animals all carry them about. Aerially applied sprays commonly reach the target in amounts equal to less than 50 percent of the quantity distributed (Cope and Bridges, 1963; Hitchcock, 1965; Pillmore and Finley, 1963).

Drift from the intended site has been shown both by residue analyses and by animal mortality. For example, organochlorines were unintentionally carried by air to forage and food crops in certain areas (U.S. Department of Agriculture, 1962). Dieldrin drifted to nearby pastures in quantities sufficient to kill sheep and cause illness of cattle (Luckmann

Table 1.--Domestic disappearance and production of organochlorine pesticides in the United States, 1949 to 1966

[U.S. Department of Agriculture (1953-1967). Disappearance calculated as amount manufactured less inventories less exports. Domestic disappearance of aldrin group for 1965-66 supplied through courtesy of H. H. Shepard, Agricultural Stabilization and Conservation Services]

Year[1]	Domestic disappearance (thousands of pounds)		Production (thousands of pounds)	
	Aldrin group[2]	DDT	Aldrin group[2]	DDT
1965-66	86,646	45,603	130,470	141,349
1964-65	80,568	52,986	118,832	140,785
1963-64	83,161	50,542	105,296	123,709
1962-63	79,275	61,165	105,986	178,913
1961-62	82,125	67,245	106,276	167,032
1960-61	78,260	64,068	103,763	171,438
1959-60	75,766	70,146	90,671	164,180
1958-59	73,331	78,682	86,868	156,741
1957-58	78,834	66,700	98,280	145,328
1956-57	52,500	71,000	75,424	124,545
1955-56	61,570	75,000	86,659	137,659
1954-55	54,400	61,800	77,025	129,693
1953-54	35,420	45,117	45,169	97,198
1952-53	34,050	62,500	29,000	84,366
1951-52	--	70,074	49,000	99,929
1950-51	--	72,688	--	106,139
1949-50	--	57,638	--	78,150
1948-49	--	--	--	37,904

[1]Domestic disappearance is computed for the production year and spans two calendar years. Production is computed for the calendar year, which is the second of the two years listed.

[2]Aldrin group includes aldrin, chlordane, dieldrin, endrin, haptachlor, and toxaphene.

and Decker, 1960). Airborne pesticides from cottonfield treatments killed large numbers of fish, snakes, turtles, frogs, and several egrets (Boyd et al., 1963).

Even after pesticides reach the target, part of them may leave again by various routes, for example, by codistillation with water (Acree et al., 1963; Bowman et al., 1964) and volatilization from soil surfaces (Barthel et al., 1960; Bowman et al., 1964; Harris and Lichtenstein, 1961; Young and Rawlins, 1958). The importance of evaporation as a factor influencing persistence of organochlorine residues on crops has been emphasized by Decker (1966). Organochlorines ordinarily move only short distances in soils by leaching (Lichtenstein, 1958), but heavy rains or flooding can carry away significant amounts with the surface soil. Runoff from cottonfields during heavy rains produced heavy fish kills in 15 northern Alabama streams, apparently eradicating all fish life from some of them (Young and Nicholson, 1951).

In one area treated with dieldrin at 4.7 pounds per acre, runoff from the first rain killed minnows even when the runoff water was diluted 3 to 1 with clean water. Runoff from the third rain killed a little less than 50 percent of the test fish, and the runoff from the fourth rain still produced some mortality (Tarzwell and Henderson, 1957).

Circulation of pesticides in irrigation systems is important in certain areas. In the Tule Lake-Klamath drainage area of Oregon and California, for example, more than 1,000 fish-eating birds were killed in the 4 years from 1960 through 1963, apparently by toxaphene in the drainage and irrigation waters of an essentially closed system (Keith, J. O., 1964).

Loss of pesticides from soils by translocation into plants is an interesting though probably quantitatively minor route (Lichtenstein, 1959; Lichtenstein and Schultz, 1960, 1965; Westlake and San Antonio, 1960).

Despite these various routes of loss, substantial residues of organochlorine may persist in soil for months or years (Edwards, 1966; Lichtenstein, 1965; Lichtenstein and Polivka, 1959; Lichtenstein and Schulz, 1959; Nash and Woolson, 1967).

OCCURRENCE OF RESIDUES: RECORDS AND SURVEYS

Most living organisms now contain organochlorine residues. This has been verified by a tremendous sampling and analytical effort, which still continues.

Residues in Wild Animals

Organochlorine residues occur in almost all birds analyzed in many countries. Birds found

dead in coastal habitats of the Netherlands contained up to 9 parts per million of dieldrin plus endrin in the breast muscle and up to 0.7 ppm of DDE. Muscle tissue of dead or dying birds of prey contained 0.4-30.0 ppm of dieldrin, 1.2-80.0 ppm of DDT and its metabolites, and usually also heptachlor epoxide and lindane (Koeman and van Genderen, 1965). Eggs of seven species of Australian birds contained both DDE (0.6-12.0 ppm) and dieldrin (trace to 0.02 ppm) (Butcher, 1965). Birds of prey, gamebirds, and seabirds in Ireland contained aldrin, dieldrin, lindane, DDT, and mercury. Quantities in eggs were small (less than 1 ppm); amounts in tissues were similarly low, except in pheasants and pigeons that were obvious victims of pesticide poisoning (Eades, 1966). Antarctic animals collected early in 1964 (Sladen et al., 1966) and others collected a year later (George and Frear, 1966) contained DDT and its metabolites. Additional material collected later (Tatton and Ruzicka, 1967) was subjected to exhaustive analytical studies that confirmed the identifications.

Residues in California birds and fish have been studied extensively (Hunt, 1964; Keith, J. O., and Hunt, 1966). Nearly all specimens collected in a pesticide survey in 1963 contained residues (Hunt, 1964). Samples contained, DDT, DDD, DDE, dieldrin, endrin, heptachlor epoxide, toxaphene, benzene hexachloride, and chlordane, usually several kinds in one individual. In fat from 128 striped bass from six localities, residues of DDT and its metabolites ranged from 2 to 124 ppm and averaged 64 ppm. In the same samples, dieldrin averaged 0.8 ppm and endrin 0.1 ppm. In Connecticut woodland areas where no spraying had been done, fish, shellfish, birds, and mice all contained DDT (Turner, 1965).

Living insects of 11 species sampled throughout Louisiana contained from 0.1 to as much as 90 ppm of DDE and up to 0.5 ppm of endrin and 0.2 ppm of dieldrin (Atallah and Nettles, 1966; Newsom, 1967).

Shellfish samples from eight States in various parts of the country contained DDT + DDE residues ranging from less than 0.008 ppm to 0.9 ppm (Campbell et al., 1965).

Residues in edible game have been the subject of a number of studies and surveys.[1] The fat of big game from untreated areas, and from statewide surveys that include many untreated areas, contains small quantities of DDT, 0.2 ppm or less on the average. Summer treatments of forest areas with 3/4 - 1 pound of DDT per acre may result in high residues in the game in the fall, but do not always do so. In one study residues in fat averaged 7-12 ppm (up to 43 ppm), but in another they averaged only 3 ppm (up to 11 ppm).

Residues in upland game birds may be high. Following application of 1/2 pound of DDT per acre, residues in fat of blue grouse averaged 46 ppm the first season (up to 280 ppm) and persisted at an average of 22 ppm (up to 110 ppm) in the second season. Two samples taken in the third season had 15 and 21 ppm. Pheasants in California's Sacramento Valley contained an average of 123 ppm of DDT and its metabolites in fat (up to 5,448 ppm) plus an average of 0.8 ppm of dieldrin. Residues in pheasants from other parts of California were lower.

Woodcocks collected in Michigan in 1963 contained about 2 ppm of DDE and 5 ppm of heptachlor epoxide in the carcasses; residues in their fat would be at least 10 times these. Woodcocks that arrived in New Brunswick in the spring of 1963 contained 6 ppm of DDE and 5 ppm of heptachlor epoxide in the carcasses.

Ducks (mallards, teals, and scaups) collected in California statewide surveys averaged about 25 ppm of DDT and its metabolites in fat (up to 150 ppm). Oldsquaws in Lake Michigan contained even larger amounts.

Extensive sampling of mallards and black ducks from New York and New Jersey indicates fat residues averaging about 15 ppm of DDT and its metabolites plus 1 ppm of dieldrin.

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[1] A narrative summary is given here. Details and references appear in table 2.

Table 2.--Pesticide residues in wild game

Species and number	Tissue	Residue	Mean and range (ppm)	Treatment	Time from treatment	Location and date	Citation
Blue grouse (28)	Fat	DDT & metabs.	46 (1.5-280)	1/2 lb DDT/A for spruce budworm, 1963	First season	Montana, 1963, 1964 and 1965	Mussehl and Finley (1967)
Blue grouse (35)	Fat	DDT & metabs.	22 (1-110)		Second season		
Blue grouse (2)	Fat	DDT & metabs.	15,21		Third season		
Pheasant (146)	Fat	DDT & metabs.	123 (tr-5448)	Rice growing areas	--	California Sacramento Valley, 1960's	Keith and Hunt (1966)
		Dieldrin	0.8				
Woodcock (10)	Carcass	Hept. epox.	0.7 (0-2.4)	--	--	Michigan, 1960	Patuxent Wildlife Research Center (unpubl.)
Woodcock (11)	Carcass	Hept. epox.	5.0 (2.2-8.4)			Michigan, 1963	
Woodcock (22)	Carcass	DDE	0.6 (0-4.6)			Michigan, 1960	
Woodcock (11)	Carcass	DDE	1.6 (0.2-6.3)			Michigan, 1963	
Woodcock (7)	Carcass	Hept. epox.	2.3 (1.1-3.3)			Maine, 1960	
Woodcock (10)	Carcass	Hept. epox.	1.8 (0.3-3.2)			Maine, 1963	
Woodcock (13)	Carcass	DDE	1.2 (0-3.5)			Maine, 1960	
Woodcock (12)	Carcass	DDE	1.0 (0.1-3.0)			Maine, 1963	
Woodcock (10)	Carcass	Hept. epox.	4.7 (2.0-11.0)	1/4 - 1 lb DDT/A yearly from 1952	--	N. Brunswick 1963 (spring)	Wright (1965)
Woodcock (10)	Carcass	DDE	5.7 (tr-16.8)				
Woodcock (11)	Carcass	Hept. epox.	0.4 (tr-1.5)			N. Brunswick, 1961 (fall)	
Woodcock (11)	Carcass	DDE	1.7 (tr-7.0)				
Bobwhite (13)	Carcass	Hept. epox.	1.0 (nd-2.2)	2 lb Hept./A	2-3 years	Georgia	Rosene (1965)
Songbirds (5)	Carcass	Hept. epox.	32 (15-58)		1 week		
Mallard (11)	Fat	DDT & metabs. (+ tr dieldrin)	28 (tr-150)	--	--	California, Statewide, 1963-65	Keith and Hunt (1966)
Cinnamon teal (5)	Fat	DDT & metabs. (+ tr dieldrin)	23 (tr-106)				
Lesser scaup (8)	Fat	DDT & metabs.	28 (1-103)				
Old squaw ducks, adults (2)	Fat	DDT & metabs.	138 (0-285)	--	--	Lake Michigan, 1964	Hickey et al. (1966)
Old squaw ducks, juveniles (3)	Fat	DDT & metabs.	73 (35-111)				
Mallard and black ducks (18 pools of 25 wings each)	Wing bases	DDT & metabs.	1.63 (est. 16.3 lipid ppm)	--	--	New York and New Jersey, 1964-65	Heath and Prouty (1967)
		Dieldrin	0.09 (est. 0.9 lipid ppm)				
Mule deer (24)	Fat	DDT & metabs.	12 (0.05-43.0)	1 lb DDT/A 1959, 1960 and 1962	1 1/2-4 mo.	Montana, Colorado, & N. Mexico	Pillmore & Finley (1963)
Elk (23)	Fat	DDT & metabs.	7 (0-22)		3 mo.	N. Mexico	
Elk (11)	Fat	DDT & metabs.	1.4 (<0.5-3.4)		15 mo.	N. Mexico	
Elk (34) and deer (3)	Fat	DDT & metabs.	<0.7 max.	3/4 lb DDT/A, 1963 (summer)	Prespray, 1962 (fall)	Washington	Wash. Dept. Nat. Res. (1964)
Deer (12)	Fat	DDT & metabs.	2.7 av.		Postspray, 1963 (fall)		
Elk (25)	Fat	DDT & metabs.	2.99 av.		Postspray 1963 (fall)		
Big game (antelope, deer, elk, moose, Mt. goat)(341)	Fat	DDT & metabs.	<0.1 av.	Little or none since 1957	--	Idaho & Washington, 1962	Walker et al. (1965)
Mule deer (2)	Fat	DDT & metabs.	0.4-2.8	Orchards & vicinity, treatment varied	--	Colorado, 1966	Jewell (1967)
		Dieldrin	0.02-0.05				
		Endrin	tr-0.06				
White-tailed deer (23), mule deer (13), pronghorns (9), and elk (2)	Fat	DDT & metabs.	0.2 av.	--	--	S. Dak., 1964 (fall)	Greenwood et al. (1967)
		Dieldrin	tr in some				

Residue Monitoring

In the United States, a National Monitoring Program has been undertaken by the Federal Government, with the objective of providing baseline readings and a record of trends in this form of environmental contamination. Residues are monitored in foods and feed, in people, in fish, wildlife, and estuaries, in water, and in soil. This program is described in the Pesticides Monitoring Journal (vol. 1, no. 1, June 1967). The part of the program that relates to fish, wildlife, and estuaries is described by Dustman (1966) and by Johnson, R. E., et al. (1967). Samples of fish (carp, buffalo, black bass, channel catfish, green sunfish, yellow perch, rainbow trout, or squawfish) are taken twice a year at 50 freshwater collection sites scattered throughout the major drainage systems of the country; samples of oysters (or clams) and bottom sediments, three composited samples of each, are taken from 24 major estuarine systems along the Atlantic, Pacific, and Gulf Coasts; approximately 500 composite samples of mallard (or black) duck wings are assembled from ducks shot during the hunting season in each of the 48 contiguous States; and approximately 50 composite samples of starlings are collected twice a year at randomly selected locations throughout the country. Bald and golden eagles found dead are also included in the program, although the numbers and locations are variable.

In June 1966, the Organization for Economic Cooperation and Development sponsored a meeting in France on the unintended occurrence of pesticides in the environment. At this meeting the decision was reached to explore the feasibility and desirability of standardized international sampling. Results of the first sampling were reported at a meeting in Scotland in September 1967 under the sponsorship of the OECD and the Natural Environmental Research Council of the United Kingdom, and plans were made for further study and comparisons.

These national and international monitoring efforts indicate that the need to know the kinds and quantities of organochlorine pesticides in the environment is widely recognized.

OCCURRENCE OF RESIDUES: PATTERNS OF DISTRIBUTION

Residues in Birds Related to Food Habits

Quantities of pesticide in the tissues of wild birds are related to their food habits, and presumably are primarily a reflection of contamination of the food supply. Differences in abilities of species to absorb and metabolize pesticides may produce differences in residue accumulation that cannot be fully separated from differences due to food habits.

Raptorial and fish-eating birds in Britain had considerably higher organochlorine residues than did herbivores (Cramp et al., 1964; Moore and Walker, 1964; Ratcliffe, 1965; Cramp and Olney, 1967). Eggs of peregrine falcons, great crested grebes, and herons contained residues in concentrations that were hundreds of times those in eggs of the plant-eating Canada goose and the omnivorous pheasant and carrion crow (Moore and Walker, 1964; Walker et al., 1967). Eggs of herons and grebes, which are freshwater fisheaters, contained residues in concentrations that were tens of times those in the eggs of seabirds (Moore and Tatton, 1965). Since there is little or no spraying of coastal waters with insecticides in Britain, the residues must derive either from the contamination of rivers or from aerial drift.

Alaskan peregrines, which feed primarily on birds, contained an average of 95 ppm (dry weight basis) of DDT and its metabolites in muscle, far higher residues than in small birds in the same area. Among the small birds, the migrant sandpipers, wetland feeders, contained an average of 6 ppm (dry weight basis) of DDT and metabolites in their bodies,

whereas the seed-eating migrants contained 0.7 ppm and the insectivorous migrants contained 1.4 ppm. However, even the resident boreal birds contained DDT residues averaging 0.6 ppm, similar to those of the migrant seed-eaters (Cade et al., 1968).

Scaups, which feed more heavily upon animal material, accumulated residues that were two to four times those accumulated by mallards when both were placed on a DDT-treated marsh for the same periods of time (Dindal, 1967; Dindal and Peterle, 1968).

Herring gulls in Lake Michigan contained very high residues of DDT and its metabolites by comparison with oldsquaw ducks in the same area, and considerably higher residues than did ring-billed gulls (Hickey et al., 1966). The quantities were an average of 99 ppm in breast muscle of adult herring gulls, 28 ppm in ring-billed gulls, and 6 ppm in the ducks. Food habits, history of exposure, and physiology all could have influenced the readings.

Residues in Birds of Different Countries

Residues in birds of different countries can be compared for a few species that have similar habits. Herons in Great Britain (Moore and Walker, 1964) contain residues of the same magnitude as those in bald eagles and brown pelicans in the United States (Stickel, L. F., et al., 1966a; Stickel, L. F., unpublished). Eggs of ospreys in Maryland contained about 3 ppm of DDT and its metabolites, those in Connecticut about 5 ppm (Ames, 1966), averages similar to those in eggs of British estaurine birds. Eggs of California shore birds and fisheaters contained similar amounts if allowance is made for the fact that analyses were of yolk alone rather than of yolk plus white (Keith and Hunt, 1966).

Four bald eagle eggs collected in Maine, New Jersey, and Florida in 1964-65 contained 6-16 ppm of DDT and its metabolites and 0.5-1.0 ppm of dieldrin (Stickel, L. F., et al., 1966a), amounts similar to those in 12 British peregrine eggs, which averaged 12.4 ppm DDE, 0.6 ppm dieldrin, and 1.4 ppm of other organochlorines (Moore and Walker, 1964). Eggs of Scottish golden eagles contained 0.5-7.0 ppm of dieldrin (Lockie and Ratcliffe, 1964). Two eggs of Alaskan peregrines contained about 11 ppm of DDT and its metabolites and about 0.5 ppm of dieldrin (assuming 80 percent moisture content) (Cade et al., 1968).

Eggs of cormorants nesting on interior lakes of Wisconsin, Minnesota, North Dakota, Manitoba, and Saskatchewan contained 11 ppm of organochlorine residues, primarily DDT and its metabolites but including also about 0.2 ppm of dieldrin; some also contained traces of heptachlor epoxide. White pelican eggs from the same colonies contained smaller amounts, an average of 2.4 ppm of organochlorines, including about 0.15 ppm of dieldrin (Anderson, 1967; U.S. Bureau of Sport Fisheries and Wildlife, 1967).

PESTICIDE KINETICS: GAIN AND LOSS IN THE ECOSYSTEM

Any segment of the ecosystem--marshland, pond, forest, or field--receives various amounts and kinds of pesticides at irregular and largely unknown intervals. The different animals and plants absorb, detoxify, store, and excrete the toxicants at different rates. Different degrees of magnification of pesticide residues by different living organisms in an environment hence are the practical result of many interactions that are far more complex than implied by the statement of magnification up the food chain.

The Aquatic Environment

In aquatic systems, the greatest magnification of organochlorine residues is physical, for the low solubility of most of these compounds in water and their adsorbent properties

lead to concentrations of residues in bottom mud that may reach millions of times the concentrations in the water (Johnson, W. D., et al., 1967).

Filter feeders, such as oysters, pass large quantities of water through their systems, extracting flocculent food particles and organochlorine pesticides. A 10-day exposure to a mixture of eight pesticides in the water at 0.001-0.05 ppm resulted in tissue concentrations of 1-289 ppm. Concentration factors varied from 60 times for lindane to 17,600 times for heptachlor. The concentration factor for the DDT group was 15,000 (Wilson, 1966).

Oysters thus provide a sensitive reflection of changes in the environment, as was shown in a practical example (Butler, 1966a). In October, DDT residues in oysters from a Florida reef suddenly increased fourfold, and the high level continued through December. The following year, the investigators learned that residues in planktonic organisms upon which oysters feed increased markedly in the summer, and residues in oysters again increased in the fall. The source of residues was traced to July-August spraying of DDT on seaweed windrows for control of larvae of the fly Stomoxys calcitrans.

In a tidal marsh ditch in Florida, treated with 0.2 pound of DDT per acre, surface water samples contained 0.03 to 0.04 ppm. Sediment samples reached 3.4 ppm (dry weight); vegetation samples reached 75 ppm (dry weight); and living fish of five species accumulated 4 to 58 ppm (wet weight) of DDT (Croker and Wilson, 1965).

In a farm pond, DDT in the water at 0.02 ppm produced a maximum concentration of 8 ppm in bottom mud in 1 day, a maximum of 31 ppm in or on vegetation in half an hour, a maximum of 2 ppm in crayfish in 3 weeks, and 2 to 4 ppm in trout and bullhead; amounts in fish were not greatly different in samples taken at different times, from 1 week to 16 months after treatment (Bridges et al., 1963).

In the Green Bay area of Lake Michigan, mud samples contained 0.014 ppm of DDT and its metabolites; crustaceans that were a principal fish food contained 0.41 ppm (Hickey et al., 1966). Alewives taken from herring gulls averaged 3.4 ppm. Twelve seemingly healthy herring gulls, collected on their nesting islands, had 99 ppm of DDT and its metabolites in breast muscle and 2,441 ppm in fat.

Among the inhabitants of a Long Island marsh treated annually with DDT for many years, birds contained the highest residues (3-26 ppm, with a median of 5 ppm in whole bodies of 16 birds of seven species; 76 ppm in one ring-billed gull) (Woodwell et al., 1967). Fish of eight species contained 0.17 - 1.33 ppm with a median of 1 ppm, and plankton contained 0.04 ppm.

On the Tule Lake refuges in the Far West, organochlorines adhered to suspended organic matter in the water, and from there they apparently entered the living food chain by way of small invertebrate animals which fed upon the suspended material (Keith, 1966b). Where invertebrates were abundant, relatively small quantities of organochlorines remained to settle out, and concentrations on bottom sediments were lower than on floating material. The opposite was true where invertebrates were scarce and nearly the full residue burden of the floating material reached the bottom; sediment levels then were several times the levels on suspended material. Plants also accumulated smaller amounts of organochlorines in waters where invertebrates were abundant than in waters where invertebrates were absent.

Analyses of birds, fish, and invertebrates from Pacific Ocean localities showed that DDT is also a component of marine ecosystems and that pelagic species may accumulate high concentrations of DDT residues (Risebrough et al., 1967). As in other studies, residues were generally higher in birds than in fish and higher in fish than in invertebrates. Among the birds, residue levels in native California species were higher than those in the northern migrants.

In a 4-acre marsh in Ohio, DDT (labeled with radioisotopes) appeared in significant quantities in tadpoles, small carp, bladderwort,

and lesser duckweed within 4 hours of treatment at the rate of 0.2 pound per acre. The highest concentration was 245 ppm in algae in 3 days (Peterle, 1966; Meeks, 1968). Plants and most invertebrates accumulated their highest residues during the first week, while DDT could still be detected in the water. Residue curves in plants and water almost mirrored each other during the first 2 weeks, declining in both, suggesting that the plant residues of DDT were largely a surface phenomenon at that time.

In the marine environment, certain bacteria have exhibited a remarkable ability to accumulate DDT (Johnson, R. F., 1967).

Typical concentration of DDT and dieldrin residues was reported for two Pennsylvania streams and their watersheds prior to any known treatments of the area with these materials (Cole et al., 1967). For example, residues in watershed soils and stream sediments were quite similar, generally occurring at a few parts per billion, while residues in brook trout (Salvelinus fontinalis) were approximately 20-100 times those of the stream sediments; residues in white suckers (Catostomus commersoni) were 6-15 times those in the trout.

In marine samples taken off the shores of Great Britain, residues of DDE and dieldrin in species of different trophic levels generally increased in the expected order, although there were exceptions within certain individual food chains that suggested the influence of physiological factors (Robinson et al., 1967a).

Relations between residue concentrations in different environmental components have been shown in many other studies, for example: DDT in water, mud, algae, and fish in New York State (Mack et al., 1964); DDT in water, mud, flora, and fauna in Utah (Warnick et al., 1966); toxaphene in water, mud, aquatic plants, aquatic invertebrates, trout, and salmon in Oregon (Terriere et al., 1966); aldrin and dieldrin in soils, water, and crawfish in Louisiana (Hendrick et al., 1966).

Although pesticide residues cannot disappear from the ecosystem short of complete degradation, they may be lost from individual components. The decline of residues from individual portions of the environment has been shown in field studies. For example: In a farm pond, treated with DDT at the rate of 0.02 ppm, residues in mud declined to pretreatment levels in 8 weeks; residues on and in vegetation declined rapidly within a day, but did not appear greatly different in 1-week and 8-week posttreatment samples. New vegetation arising a year later, however, had residues similar to the pretreatment sample. Even after 1 1/4 years, residues in trout were still essentially the same as they were soon after treatment, and residues in bullhead declined little if any in the same period (Bridges et al., 1963).

DDT residues in fish of the Farmington and Connecticut River watersheds declined significantly from the fall of 1963 to the spring of 1964 (Tompkins, 1964).

The Terrestrial Environment

Filter feeders in the terrestrial environment, such as earthworms, cannot rival the oyster in concentrating residues, and the process is slower, matching the slow passage of soil through the digestive tract. In DDT-sprayed elm environments in Wisconsin, for example, pesticide residues were 19 ppm (dry weight) in the top inch of soil, whereas earthworms from the same soil contained 157 ppm (dry weight), about eight times as much (Hunt, 1965). In one area in Illinois where DDT was used for Dutch elm disease control, fallen autumn leaves from sprayed trees contained 20 to 28 ppm of DDT, and the soil beneath contained 11 to 18 ppm (dry weight) (Barker 1958). Earthworms living in the soil and coming to the surface to eat the leaves accumulated 4 to 194 ppm in various body tissues, 403 ppm in crop and gizzard.

Earthworms from 67 fields of 14 crop types in eight States concentrated organochlorines to an average of nine times the concentration in the surrounding soil (U.S. Bureau of Sport Fisheries and Wildlife, 1967; data of C. D. Gish). Earthworms from two Missouri fields treated with aldrin for 16 or 15 of the past 17 years contained 3.5 and 4.0 ppm (dry weight) of aldrin and dieldrin, concentrations 11 and

8 times those found in the soil (U.S. Bureau of Sport Fisheries and Wildlife, 1968; data of L. Korschgen). Crickets contained smaller amounts, 0.5 and 0.7 ppm. Ground beetles of one species contained 1.0 and 2.6 ppm, whereas those of another species contained 7.0 and 6.8 ppm. Residues were highest during midsummer when young birds would rely upon them most heavily for sustenance. In August, for example, residues in one of the kinds of ground beetles averaged 21 and 16 ppm of dieldrin in the two fields (all in this paragraph on a dry weight basis).

Slugs in a cottonfield accumulated 18 times and earthworms 11 times the soil residues of organochlorines (U.S. Bureau of Sport Fisheries and Wildlife, 1966, 1967; data of C. D. Gish). The slugs contained 53 ppm of DDT and its metabolites, 0.4 ppm of dieldrin, and 1 ppm of endrin; earthworms contained 32 ppm of DDT and its metabolites and traces of dieldrin and endrin (all in this paragraph on a dry weight basis).

Residues of BHC and DDT in soils, soil invertebrates, and vegetation of a British apple orchard varied seasonally in relation to treatments (Cramp and Olney, 1967). Total soil residues were highest in May, following a March treatment, and lowest in September despite two intervening treatments. The reason was apparent in the high residues on the leaves and grass in the summer sampling. Over a larger area of Britain, residues of dieldrin and DDT plus metabolites also appeared to vary seasonally in accordance with the pattern of use of these materials.

Microorganisms also have been shown to accumulate DDT and dieldrin from culture media and from soils (Chacko and Lockwood, 1967; Ko, 1967).

Plants absorb pesticides from the soil, and the concentrations in edible seeds sometimes are a substantial part of the soil concentrations. For example, soybean seeds contained dieldrin or heptachlor epoxide in concentrations about 10 percent of those in the soil; corn and barley contained less than 1 percent of the soil concentration. Peanuts stored a larger proportion, but still less than half the concentration in the soil (Bruce et al., 1966). In another study, peanuts stored 4-7 times the soil concentration of heptachlor and 2-4 times the concentration of aldrin plus dieldrin (Beck et al., 1962).

Plants may acquire organochlorine residues from the surrounding air. This was shown in an experiment in Great Britain where leaves of kale kept in containers on plots treated with aldrin, but not in contact with the soil, contained 0.04 ppm dieldrin on low-dosage plots and 0.2 ppm on high-dosage plots (Walker 1966a).

Following DDT treatments at 3/4 pound per acre for white-fir sawfly control in California, residues gradually declined in some components of the environment, but increased in others (Keith and Flickinger, 1965). Residues in litter and vegetation declined steadily from the time of treatment. Initial posttreatment levels were 7 to 20 ppm. After 6 weeks 8 to 36 percent remained, and after 3 months 2 to 32 percent remained. Residues were most persistent in litter, least so in sagebrush. Residues in robins were at or below pretreatment levels in 3 months; residues in flycatchers and tanagers were essentially the same after 6 weeks as they were soon after treatment. Juncos and chickadees appeared to be subjected to a real exposure to DDT, for after 3 months they had 6.0 and 11.7 ppm of DDT residues, 231 percent and 638 percent of the early posttreatment levels. Insects sampled on treatment day contained 206 ppm; those taken 1 to 2 days after treatment contained 84 ppm; and those taken alive after 1 month contained 2 ppm.

In Great Britain, studies have been initiated in pine woodland treated (for the first time) with 1 pound of DDT per acre to determine residues in various parts of the environment and their changes with time. Parallel studies were undertaken in areas treated with DDT in 1954. Early results are reported by Walker (1966b).

Animals absorb organochlorines from their food and store them in their tissues; they also metabolize[2] these chemicals and excrete them. It is generally assumed that an equilibrium, or steady state, will be reached at continuous low levels of intake so that, in time, losses will balance gains and the body content will remain approximately the same.

This is an oversimplified statement of the end result of many complex biochemical events in an interrelated system involving various tissues and organs of the body in each of which processes of gain and loss occur. Underlying principles have been recognized and applied extensively in radioecology and in pharmacology, and mathematical models have been presented (for example, from the pharmacological viewpoint: Nelson, 1961; De Jonge, 1961).

Experimental Studies

MAMMALS

Experimental studies with mammals soon established the likelihood that pesticide kinetics could be described according to the principles of pharmacodynamics. These earlier studies are summarized by Hayes (1959), Negherbon (1959), and Rudd and Genelly (1956). There have been many studies since, in which rates and other mathematical relationships are considered in more detail. These studies are reviewed by Hayes (1965).

It appears to be a general rule that residues of organochlorines are lost from the body after the toxicant is removed from the diet--suggesting that equilibrium levels would be attained in long-term studies (the numerous papers demonstrating this loss will not be cited here), but Hayes (1965) points out that there are few such long-term demonstrations, even for DDT. Quaife et al. (1967) reviewed experimental studies of aldrin and dieldrin in relation to the equilibrium concept and concluded that there have been no fully adequate studies to demonstrate that saturation levels of dieldrin in the fat can be reached and maintained for a significant part of the animal's lifetime. However, there have been studies in which organochlorines were fed long enough that an apparent equilibrium was achieved, and loss rates were established for a subsequent period when toxicant was removed from the diet. For example, Laug et al. (1950) demonstrated a storage peak at 19 weeks in tissues of rats fed DDT, and a subsequent decline in tissue content when DDT was removed from the diet. Ludwig et al. (1964) reported similar results for aldrin fed to rats for 12 weeks; a balance was achieved at about 8 weeks, and residues declined after dosage was discontinued. Williams et al. (1964) showed increase in residue content to an apparent equilibrium and postdosage loss of five pesticides (heptachlor epoxide, dieldrin, endrin, lindane, and DDT) in the milk of cattle.

BIRDS

Chickens fed a combination of dieldrin, endrin, lindane, heptachlor epoxide, and DDT for 96 days showed an apparent equilibrium in eggs and tissues within 2 or 3 months, and a decline in residues when pesticides were no longer fed (Cummings et al., 1966, 1967). In contrast, residues in eggs of two chickens fed aldrin or dieldrin in their diets increased continuously for more than 300 days, then declined to lower levels during the next 400 days although dosage continued (Brown et al., 1965). Many other papers show depletion of organochlorine residues from domestic birds after dosage is discontinued. The principles of gain and loss of organochlorine residues apply also to wild species of birds, although studies are fewer and less comprehensive.

Bald eagles experimentally fed diets containing 5 ppm (dry weight) of DDT accumulated residues of DDT, DDD, and DDE in their tissues during dosage periods of 2 and 4 months (Stickel, L. F., et al., 1966a). Residues

[2] Chemical change of organochlorine pesticides, both within and outside living organisms, is the subject of a voluminous literature which is not reviewed in the present paper.

were considerably higher at 4 months than at 2 months, showing that equilibrium had not been reached at 2 months. Residues of DDT and DDD declined in tissues of birds fed untreated food for 2 months after being fed DDT-treated food for 2 months. Residues of DDE, however, did not decline demonstrably in that time. Approximations of rates of gain and loss of DDT were similar to those reported for other species.

Loss of residues from tissues of various wild species has been suggested in other studies: house sparrows, DDT (Bernard, 1963); male cowbirds, DDT (Stickel, L. F., et al., 1966b); starlings, DDT (Harvey, 1967); grackles, DDT (Walley, 1965); woodcock, heptachlor epoxide (Stickel et al., 1965); pheasant eggs, dieldrin (Lamb et al., 1967).

FISH AND SHELLFISH

Oysters have a great capacity for accumulation of residues, but when they are placed in uncontaminated circulating sea water, the residues decline at regular rates, depending on the initial concentration. Oysters having a body burden of 150 ppm of DDT may lose two-thirds of it in 50 days, but require another 40 days before residue levels decrease to 6 ppm (Butler, 1966b).

Fish may accumulate residues both from the food they eat and from the water that surrounds them (Holden, 1962; Allison et al., 1964; Andrews et al., 1966). Under laboratory conditions, in a continous flow system where pesticide concentrations in water were kept constant, fathead minnows (Pimephales promelus) exposed to water containing 0.000015 ppm of endrin had total body concentrations 10,000 times those in the water (Mount and Putnicki, 1966). Croakers (Micropogon undulatus exposed for 5 weeks to 0.0001 ppm of DDT in water accumulated concentrations 20,000 times these (Hansen, 1966). Trout (Salmo gairdneri) that died after 17-23 days of exposure to water containing dieldrin at 0.0023 ppm had muscle residues 3,300 times the water concentration (Holden, 1966).

Some studies of fish have been carried to an apparent equilibrium. In one experiment, pinfish (Lagodon rhomboides) exposed to 0.001 ppm of DDT in the water reached a steady state in 2 weeks; body residues at this time were 12 ppm, a concentration 12,000 times that in the water (Hansen, 1966). Pinfish exposed to a smaller amount of DDT, 0.0001 ppm, reached a steady state in approximately the same length of time; body residues were 4 ppm, a concentration 40,000 times that in the water.

Cutthroat trout (Salmo clarki lewisi) exposed once monthly to a 30-minute bath in water containing 0.3 and 1.0 ppm of DDT (amounts that eventually resulted in considerable mortality) contained 4 ppm after six treatments; residues fluctuated between 3 and 6 ppm during the next 12 treatments (Allison et al., 1964). Trout exposed to 0.03 and 0.01 ppm contained residues that followed a similar pattern, but apparently reached maxima somewhat more slowly. Trout fed 0.3 mg/kg (a sublethal dose) in their diet once each week accumulated 4 ppm after approximately 49 feedings; residues remained at 3-4 ppm through 79 feedings. Residues in fish fed toxic doses accumulated rapidly to high levels.

Problems in Extrapolation

Experimental studies indicate similar patterns of gain and loss of organochlorine residues by many different kinds of animals. The rates differ according to the kind of chemical and the kind of animal, as would be expected. However, these differences are subject to experimental study, and pose relatively well defined problems. Variability in physiological condition and in exposure (dose) are far more serious deterrents to extrapolation of laboratory results to the field.

Physiological effects on residue kinetics have been shown in certain studies. For example, in one study, reduction of quantity of food altered the rate of excretion of DDT in rats (Dale et al., 1962). In another study, woodcock that had lost weight while being fed reduced rations were then given food containing toxicant for 5 days; during this time, they ate large quantities of food and accumulated heptachlor epoxide in their tissues in concentrations approaching those in normal-weight

birds fed ad libitum for much longer times, up to 52 days (Stickel, W. H., et al., 1965).

Recurrence of toxic symptoms, and mortality long after exposure has ceased, suggest that kinetic changes have occurred, perhaps triggered by weight loss or other stress (examples of these occurrences are included in a later section). Seasonal fat storage, growth, disease, reproduction, and perhaps other factors, each could alter the pattern of residue increase and loss predictable in adult animals exposed uniformly to continuous low doses of toxicant.

The experimental studies of Andrews et al. (1966) on the effects of heptachlor on bluegills reveal that even simulated natural environments present the opportunity for variations and interactions that affect residue concentrations in unpredictable ways.

As a further complication, field exposure is rarely uniform or continuous, and the toxicants are not presented singly. The mixtures of pollutants to which wild animals are exposed run the whole gamut from oil to heavy metals. Organochlorine pesticides represent only one of many chemical contaminants.

PESTICIDE EFFECTS: MORTALITY

Field Observations

Pesticides are distributed for the purpose of inducing population changes, an objective usually achieved. Immediate mortality of target organisms is easily observed in fact and in effect. Perforce, organisms whose demise was not intended die also.

On numerous occasions birds, fish, and sometimes mammals, have died in conspicuous numbers following pesticide treatment programs (Bender, 1957; Cramp and Conder, 1961, 1965; Schneider, 1966; Przygodda, 1966; Cramp et al., 1962, 1963, 1964; Dustman and Stickel, 1966). Records of birds dropping from the air in flight have appeared in the literature of several countries (Butler, 1966a, and in correspondence; Carnaghan and Blaxland, 1957; Keith, J. O., 1966; Koeman and van Genderen, 1965; Cramp and Conder, 1961).

Wildlife mortality in the United States has been a common aftermath of recommended treatment practices, as shown in table 3. However, accidents and carelessness continuously accompany pesticide use, at least on a percentage basis, and are a part of the problem.

The continuing nature of the problem is shown by the annual lists of pollution-caused fish kills in the United States (U.S. Department of Health, Education, and Welfare, 1960-1964; U.S. Department of the Interior, 1965-1966). The number of incidents due to agricultural poisons varied from 49 to 93 per year during the report period; average kills per incident were 2,000 to 128,000 fish. Reporting probably is not altogether uniform, nor are all pesticide-caused fish kills included, especially smaller ones. Accidental pollution from pesticide manufacturing plants are not separated in the reports from other kinds of industrially caused fish kills, but they, too, contribute to the problem.

Outright mortality must be included with the potentially significant factors that may alter populations, especially of the scarcer or less adaptable species.

Residue Interpretation

Diagnosis of pesticide-induced mortality from residue content of the victims has been fraught with difficulty. The problem was well stated by Radeleff and Bushland (1960):

"The use of chemical or biological determinations in the diagnosis of poisoning by insecticides is vastly complicated by their behavior. I doubt that sufficient emphasis can be given to this problem. It is not readily understood because the necessary explanations

Table 3.--Effects of organochlorine pesticides on wild animals

[Table from Dustman and Stickel (1966), which contains documentation]

Chemical	Rate	Purpose	Location	Effect
Aldrin	--	Rice seed protection	Texas	Widespread mortality of fulvous tree ducks.
Aldrin	2 lbs./A	Japanese bettle control	Illinois	Nearly complete elimination of many species of songbirds. Heavy mortality of gamebirds. Some mortality of mammals.
DDD	50-70 ppm in water	Clear Lake gnat	California	Death of grebes and reduction of breeding population.
DDT	--	Dutch elm disease control	Maine Michigan Wisconsin New Hampshire	Heavy mortality of robins and songbirds.
DDT	--	Gypsy moth and biting flies	New York	Cessation of reproductive success of trout due to death of fry.
DDT	--	Forest protection	Connecticut	Trout kill due to food depletion.
DDT	--	Agricultural drainage	California	Death of many fish, some birds.
DDT	1/2 lb./A and 1 lb./A	Spruce budworm and blackheaded budworm	New Brunswick British Columbia	Salmon and trout populations reduced and production curtailed.
DDT	--	Rice pests	California	Some deaths of mallards, pheasants and other birds.
DDT	0.2-1.6 lb./A	Mosquito control	Florida New Jersey	Deaths of fish, crabs, frogs, lizards, and snakes.
Dieldrin	2-3 lbs./A	White-fringed bettle. Japanese bettle	Virginia Illinois	Heavy mortality of quail, songbirds and waterbirds, rabbits and some other mammals.
Dieldrin, DDT and others	--	Routine agricultural applications	California	Pheasant production reduced.
Dieldrin	1 lb./A	Sandfly larvae	Florida	Heavy fish mortality.
Endrin	0.8 lb./A	Cutworm	California	Heavy rabbit mortality.
Heptachlor or Dieldrin	2 lbs./A	Imported fire ant	Georgia Alabama	Virtual elimination of birds. Populations of quail remained depressed for at least 3 years (Georgia).
Heptachlor	2 lbs./A	Japanese bettle	Illinois	Heavy songbird mortality.
Cotton Insecticides	Drift from treated fields	Cotton insect control	Mississippi	Deaths of some rabbits, birds, snakes, fish, and frogs.
Toxaphene	--	Crop protection	California	Heavy mortality of fish-eating birds each year 1960-63.
Cotton Insecticides	Surface erosion from treated fields	Cotton insects	Alabama	Heavy fish kills in 15 streams.

are apparently contradictory of our usual concept of the significance of the presence of a foreign chemical in animal or human tissues. This lack of understanding has caused us particular difficulty when toxicologists, after finding high residues, flatly state that these residues indicate death due to the material found.

"Let me carry you through some examples. In feeding tests at Kerrville with heptachlor, using a diet containing 60 p.p.m. of heptachlor, 52 p.p.m. of heptachlor epoxide could be recovered from the fat of cattle at the end of a 16-week feeding period. The cattle at that time were in excellent health and condition. . . .

"By contrast, a calf poisoned and killed by a single large dose of heptachlor, revealed a residue of only 2 p.p.m. in its fat.

"In our feeding trials with lindane, using a level of 100 p.p.m. in the feed for 10 weeks, a residue of 100 p.p.m. existed in the fat at the end of the feeding period. This was reduced to 50 p.p.m. in 4 weeks on control feed.

"By contrast, three cattle of similar breed, age, and condition, deliberately poisoned by high concentrations of lindane in dips, revealed only 23 p.p.m. 1 week later in their fat."

The same uncomfortably anomalous results appeared in examinations of other species. Nevertheless, published papers continued to report the residue content of various tissues or whole bodies of animals that died of pesticide poisoning and to view them as diagnostic, without comparisons with similarly exposed survivors.

Fortunately, the problem could be subjected to experimental study, and residues in some tissues proved to lend themselves to diagnostic interpretation, whereas those in others did not. Residues in the brain indicative of DDT poisoning have been determined, and these were similar across a wide range of animal species (Bernard, 1963; Dale et al., 1963; Stickel, L. F., et al., 1966b; Stickel, L. F., and Stickel, 1968). Experiments also have been conducted to determined critical levels of certain of the DDT metabolites (W. H. Stickel, unpublished).

Residues of dieldrin in brain that are diagnostic of hazard also have been determined and these also apply across a wide range of animal species (Stickel, L. F., et al., 1966a; Robinson et al., 1967b; Stickel, W. H., et al., 1968).

Diagnostic levels of endrin in fish blood have been established by Mount et al. (1966), and experiments with endrin and birds have been partially completed (W. H. Stickel, unpublished). The necessity for determination of diagnostic tissues was further emphasized by Butler (1966a), reporting that croakers and brown shrimp killed in the laboratory by diets of oyster containing 2 ppm of DDT uniformly showed DDT residues lower than those frequently observed in apparently healthy specimens.

PESTICIDE EFFECTS: POPULATION CHANGES

Long-lasting and ecologically significant changes in populations are less conspicuous in the early stages and the causes are difficult to ascertain. Pesticide-induced population changes have occurred among insects (for example, Bridges and Andrews, 1961; Cone, 1963; Frey, 1961; Hitchcock, 1965; Hynes and Williams, 1962; Morris, 1963; Ripper, 1956; Hopkins et al., 1966), and similar disturbances of soil organisms have been reported (for example, Edwards, 1965; Kuhnelt, 1963; Sheals, 1955).

Population effects among vertebrates also have been related to pesticide applications. In New York, DDT treatments resulted in complete elimination of trout production in certain lakes, and great reduction in others (Burdick et al., 1964). Fry died at the time of final absorption of the yolk sac.

Georgia quail populations declined strikingly soon after the land was treated with heptachlor at 2 pounds per acre; even after 3 years,

populations were still below pretreatment levels, whereas there were no measurable changes on an untreated area (Rosene, 1965).

Unwanted effects on both vertebrates and invertebrates followed a Japanese-beetle treatment program in Illinois. Large numbers of birds and mammals died following dieldrin treatments, and bird populations remained low throughout the spring and summer (Scott et al., 1959). Following this same treatment, corn borers increased 160 percent (Luckmann, 1960).

Populations of American redstarts, parula warblers, and red-eyed vireos in a bottomland forest declined 44, 40, and 28 percent over a 4-year period during which DDT was applied annually at a rate of 2 pounds per acre. Effects on the redstart were apparent immediately after treatments; effects on the parula warblers and red-eyed vireos appeared more slowly; other bird species did not show measurable decline. These three species made up a large part of the bird population, however, so that the overall decrease was 26 percent (Robbins et al., 1951).

Animals can also be affected by a reduction of their food supply. Phytoplankton communities are an important food base in aquatic environments whose productivity can be seriously affected by exposure to small amounts of pesticide. Controlled 4-hour exposure to 1.0 ppm of aldrin, chlordane, DDT, dieldrin, heptachlor, methoxychlor, or toxaphene reduced productivity 70 to 94 percent. Endrin, lindane, and mirex had smaller effects, reducing productivity 28 to 46 percent (Butler, 1963).

When a local food supply is destroyed, birds may seek food elsewhere, as has been postulated in certain studies where birds became scarcer yet no dead birds were found. Fish, however, cannot leave and so may be affected severely. Salmon hatched in the Miramichi River in New Brunswick in 1955 were reduced nearly to extinction by spraying with DDT at 1/2 pound per acre in 1956 for spruce-budworm control and the few living young were thin (Kerswill, 1957). The larger stream insects failed to return within 2 years, and these are the foods most important to the larger young salmon.

DDT applied at 1/4 pound per acre to control black-headed budworm in a forested area of southeastern Alaska resulted within 3 days in the complete annihilation of aquatic insects in two streams flowing through the area (Reed, 1966). Repopulation of stream insects was insignificant during the remainder of the summer of 1963; the insects began to approach prespray levels in the summer of 1964. Fish were not killed, but their condition (as measured by weight-length ratio) declined and continued to worsen through 1964.

A principal current concern is the possibility that organochlorines may in part be causing the dwindling reproductive success and the decline in populations of birds of prey such as eagles, ospreys, and peregrine falcons, and of estuarine species such as pelicans and egrets. Evidence includes (1) the generally higher residues of organochlorines in these species than in others, (2) the coincidence of timing of declines with large-scale treatments, and, (3) in Great Britain, an association of the decline with areas of pesticide use (Ratcliffe, 1963; Lockie and Ratcliffe, 1964; Prestt, 1965).

One of the more interesting effects on populations has been the development of resistance. Resistance among insects has become widespread, so that a continuing effort must be made to develop new methods of pest control before the present ones become ineffective.

Resistance also has developed in certain species of vertebrates. Guppies developed resistance to DDT in laboratory studies (King, 1962). Ozburn and Morrison (1962, 1964) developed a DDT-tolerant strain of laboratory mice, whose tolerance extended to lindane and dieldrin. Subsequent studies have elaborated the basis of the tolerance (Barker and Morrison, 1966). Wild rats have developed a resistance to warfarin in the field in several areas, and the resistance has proved heritable (Greaves and Ayres, 1967). Most recently, pine mice in Virginia orchards have developed resistance to endrin, long an effective control agent (Webb and Horsfall, 1967). The resistant mice

were trapped in an orchard where they had been exposed to endrin for 11 years.

Pesticide-tolerant or resistant populations of fish and possibly other coldblooded vertebrates have been shown to exist in areas of the South long subjected to pesticide treatments (Boyd et al., 1963; Ferguson et al., 1965). No top carnivores were found in nearly 100 hours' collecting in the area where the resistant fish were found (Ferguson et al., 1965), suggesting that the resistance may involve increased ability to store toxic residues, to the detriment of animals in the food chain. Local and migratory fish-eating birds could also be exposed.

In contrast, sheepshead minnows that survived DDT exposures which killed most of the fish tested tended to have offspring more sensitive to both DDT and endrin than were control fish (Holland, 1967).

PESTICIDE EFFECTS: PHYSIOLOGY AND BEHAVIOR

Many recent studies have been oriented to the discovery of the action of organochlorines in living organisms. A wide variety of physiological effects can be produced by this group of compounds, but it is not at all easy to determine whether the changes are importantly deleterious.

Little effort has been made to relate these effects to tissue residues or to evaluate physiological hazard on the basis of residues.

Enzymes and Hormones

Early investigators showed changes in liver cells of animals exposed to low doses of organochlorines (Laug et al., 1950) and these results have been confirmed more recently by electron microscopy (Ortega, 1966). Other workers sought to distinguish between liver changes that were adaptive and those that represented liver damage (Klion, 1964).

The administration of organochlorine insecticides to animals of various species stimulates both the hepatic microsomal oxidation of drugs and the microsomal hydroxylation of steroids, including androgens and estrogens (reviews by Conney et al., 1967; Durham, 1967; and Kupfer, 1967; data for fish, Buhler, 1966; data for birds, Gillett et al., 1966; Peakall, 1967). Conney et al. (1967) listed 12 such compounds. Effective amounts were small. For example, an exposure to DDT that decreased the hypnotic action of pentobarbital in rats was an amount that gave rise to the accumulation of only 10-15 ppm of DDT in fat. The effects of DDT may last 1-2 months in rats and can be produced by feeding as little as 5 ppm in the daily diet; DDT did not produce these same effects in mice, showing the importance of considering species differences (Hart and Fouts, 1965). Triolo and Coon (1966) showed that certain organochlorine compounds had a significant effect in protecting laboratory mammals against toxicity of some of the organophosphate insecticides.

Interesting aspects of multiple exposures became apparent in the discovery that DDT reduced storage of dieldrin while dieldrin increased storage of DDT (Street and Blau, 1966). Conney et al. (1967) emphasized the fact that the physiological significance of pesticide-induced increases in steroid hydroxylases in liver microsomes is not known, but suggested that, "The stimulating effect of halogenated hydrocarbon insecticides on steroid hydroxylation possibly explains the effect of these pesticides to decrease fertility in experimental animals."

Kupfer (1967) also brings out the problem of interpretation. He stated: "Attempts to correlate the effect of insecticides and other compounds on the rate of metabolism of administered steroids with the biological activity of these steroids is only preliminary. Furthermore, the physiological significance of the effects of pesticides on the metabolism of endogenous steroids is not known."

Examination of a large series of peregrine eggs collected in Great Britain between 1904 and 1950 showed a sudden and significant decrease in shell weight in the years 1946–50, without subsequent recovery (Ratcliffe, 1967). Sparrowhawk eggs followed the same pattern and time sequence. A concurrent decline was apparent in California peregrine eggs (U.S. Bureau of Sport Fisheries and Wildlife, 1968; data of J. J. Hickey and Dan Anderson).

Peakall (1967) suggested that DDT-induced enzyme action could have been involved in the reduction of shell thickness. The possibility that DDT could act directly as an estrogen is suggested by the results of studies by Welch et al. (1968).

Thus, more intensive investigation of enzymatic stimulation and estrogenic function of organochlorines is required. Studies to project results of laboratory studies to the field need particular attention. Some evidence is already available concerning the effects of organochlorines on some definable endpoints of the reproductive process.

Reproduction: Laboratory Studies

MAMMALS

Reproductive effects due to organochlorine pesticides in the diet have been shown in mammalian studies. For example, only two pups survived of 12 born to two dogs fed a daily dose of 0.2 mg/kg of aldrin with their food (approximately 3 ppm on a dietary basis) (Kitselman, 1953). Some pups appeared healthy at birth but died after a few days' nursing. Significantly fewer mice (Mus musculus) fed diets containing 200 and 300 ppm of DDT produced young than did the control mice (Bernard and Gaertner, 1964). Some adult mortality also occurred at these dosages. Laboratory mice of two strains fed diets containing 7 ppm of DDT also produced fewer litters per surviving pair than did untreated mice (Ware and Good, 1967). The differences were small and were not statistically significant. Chlordane interfered with both litter production and survival of young of rats when either sex received 320 ppm in the diet from the time of weaning (120 days) (Ambrose et al., 1953). This amount did not produce mortality during 407 days' dosage, but did reduce growth rate and produce enlarged livers. Reproductive effects were not tested at lower dosages.

Aldrin in the diet of rats at 10 or 20 ppm from 1 month of age reduced the frequency of oestrus significantly below controls. With time, the effect was overcome by adaptation in the rats fed 10 ppm, but not in those fed 20 ppm; no effect was observed in rats fed 5 ppm (Ball et al., 1953).

Dieldrin in the diet of white-tailed deer at the rate of 25 ppm reduced the reproductive rate of mature females; survival and birth weight of fawns also were reduced (Korschgen and Murphy, 1967).

Rats fed heptachlor with the daily diet at a rate of 6 mg/kg of body weight for 18 months produced smaller litters and had a higher death rate among young (Mestitzová, 1966).

BIRDS

Experimental studies have also been made of birds. An effect of DDT on spermatogenesis in chickens (Albert, 1962) and eagles (Locke et al., 1966) was produced at doses that were in the lethal range for the adults. Rubin et al. (1947) found that 310 ppm of DDT in the diet of hens lowered egg production; 620 ppm and 1,250 ppm lowered both egg production and hatchability; 2,500 ppm produced some mortality among adults. Residues in the eggs were 360 and 320 ppm at the two higher doses.

Pheasant reproductive success was reduced by dietary dosages of DDT (100 and 400 ppm), dieldrin (25 and 50 ppm), and toxaphene (100 and 300 ppm) (Genelly and Rudd, 1956). Birds were on dosage approximately 2 weeks before egg laying began. Reduction of egg production, fertility, and hatchability was apparently the result of lowered food intake and the resulting condition of the hens, and did not occur in all groups, Survival of young chicks to an age of 2 weeks was lowered in all groups and

appeared to be the critical effect. A few eggs were analyzed, with highly variable results (table 4). The importance of considering chick survival, and thus carrying a study through the critical stage of yolk absorption, was emphasized further by Koeman et al. (1967).

Reproductive success of pheasants fed DDT in their diets was studied further by Azevedo et al. (1965). Birds were fed 10 or 100 ppm in their diets for 33 days before egg laying; at that time, half were given clean food for the duration of egg laying, and half remained on dosage. Dosage of a third group, started at 500 ppm, was curtailed after 20 days because all the males and some of the females died; dosage was resumed 11 days later on a reduced basis, which consisted of 500 ppm for only 1 day of each week. A few birds on the 100 ppm dosage also died, but the dosage was not changed.

Some aspects of reproduction were affected in certain groups. Hatching success during the second half of the production period in the group receiving 100 ppm throughout was 67 percent, significantly lower than that of the controls (85 percent). Residues in egg yolks of two hens of this group averaged 125 and 133 ppm; residues in the fat of these same hens at the end of the experiment were 432 and 460 ppm.

Table 4.--Insecticide concentration in pheasant eggs

[All eggs analyzed were laid during the final week. Table from Genelly and Rudd (1956)]

ppm of chemical in total diet	ppm of chemical in eggs	Number of eggs tested
DDT		
90	162	2
355	349	4
Toxaphene		
89	54	2
243	56	4
Dieldrin		
22	3	2
42	193	3
Control		
0	0	4

Chick survival to 1 week of age was 87 percent in the 500-ppm groups, significantly lower than in the controls (98 percent). Average residues in egg yolks of four hens in these groups ranged from 263 to 473 ppm. Fat of one hen contained 1,280 ppm at the close of the study. Chick survival was 90.6 percent and 92.4 percent among the groups fed 10 and 100 ppm throughout the study, also significantly lower than controls. Average residues in 7 and 6 egg yolks of two hens of the 10 ppm group were 13 and 29 ppm; fat residues of these hens were 40 and 52 ppm at the end of egg laying.

Atkins and Linder (1967) studied reproduction of pheasant hens dosed with dieldrin once each week in capsules at rates of 2, 4, or 6 mg per week for 12 weeks. Results were presented as combined data from weeks 1 through 12. Three pheasants that received 2 mg/week of dieldrin produced eggs containing 8, 9, and 22 ppm of dieldrin in the yolks at the 11th week; egg production, fertility, and chick survival were unaffected; hatchability was increased. Three that received 4 mg/week produced eggs containing 33, 20, and 19 ppm; egg production, fertility, hatchability, and survival of chicks were unaffected. Two that received 6 mg/week produced eggs containing 52 and 46 ppm; fertility, hatchability, and survival of chicks were unaffected; egg production was reduced, presumably as a result of lowered food consumption.

Pheasants fed BHC with their diets at an average daily rate of 87 ppm reached peak egg production later than controls and produced somewhat fewer eggs (Ash and Taylor, 1964). The eggs contained residues averaging 3.4 to 12.6 ppm as dosage continued. Hatchability was similar to that of controls. Survival of chicks was not measured.

Ovulation time of Bengalese finches (Lonchura striata) was delayed progressively in relation to magnitude of DDT dosage (Jefferies, 1967). Birds received DDT with part of their diet for 6 weeks before pairing. Computed on a total diet basis, DDT dosage ranged from 11 to 54 ppm. The interval

between pairing and egg laying averaged about 16 days among untreated birds and increased progressively to an average of 25 days among birds on highest dosage, a time difference of 9 days.

Effects of several organochlorine insecticides on reproduction of quail and pheasants were presented by DeWitt (1955, 1956).

FISH

Abortion of young was observed among mosquitofish (Gambusia affinis) that survived exposure to organochlorines at dosages that killed a portion of the group (Boyd, 1964). Five to 15 percent of the survivors aborted following exposure to DDT, DDD, methoxychlor, aldrin, dieldrin, endrin, toxaphene, heptachlor, and lindane.

In another study only one of six pregnant guppies produced a second litter after being placed in water containing 0.5 or 0.75 ppb of endrin (Mount, 1962).

Mortality of cutthroat trout was high at the sac-fry stage among groups whose parents were exposed to DDT in the water (0.3 and 1.0 ppm, for 30 minutes once each month for about 15 months) or in the food (1 mg/kg once each week for about 15 months) (Allison et al., 1964). These effects were not apparent at lower dosages.

Reproduction: Field Observations

Causes of decline in reproductive success of wild animals are extremely difficult to determine with certainty. Correlation and case history--the basic techniques of natural history--must be employed, despite the pitfalls of these methods. The examples available do not all point in the same direction, but some of them at least warrant serious concern.

DDT had a conspicuous effect on trout reproduction in New York (Burdick et al., 1964). Treatments resulted in complete elimination of trout production in certain lakes, and great reduction in others. Fry died at the time of final absorption of the yolk sac, when the young fish were about ready to feed. Mortality occurred when the residue of DDT in oil extracted from the eggs was equivalent to 2.9 ppm in the fry; mortality generally increased with increased concentration of DDT; no fry survived in lots containing as much as 5 ppm, and mortality was nearly 100 percent in several lots containing considerably less.

Pheasant eggs collected from treated areas in California produced more crippled chicks than did eggs from untreated areas, and mortality of young birds also was higher in the samples from the treated area (Hunt and Keith, 1962). Egg yolks contained 106-1,020 ppm of DDT, 23-161 ppm of DDE, and 0-1.3 ppm of dieldrin. Four hen pheasants from the treated area contained 1,236-2,930 ppm of DDT, 306-717 ppm of DDE, and 0.1-25 ppm of dieldrin in their fat. These same birds contained 1.2-19 ppm of DDT, 2.3-4.4 ppm of DDE, and up to 0.3 ppm of dieldrin in breast muscle.

Herring gulls in Lake Michigan, where there was some mortality among adults due to DDT toxicity (as shown by brain residue levels), also had an unusually low reproductive success (Keith, J. A., 1966). Gull eggs contained 227 ppm of DDT and its metabolites (25 ppm of DDT plus DDD, 202 ppm of DDE); adult birds contained an average of 99 ppm in breast muscle, 2,441 ppm in fat (Hickey et al., 1966).

Graber et al. (1965) studied the nesting success of red-winged blackbirds in a 10-acre hayfield adjacent to a 20-acre wheatfield that was sprayed with 1/4 pound of dieldrin per acre. The hayfield received drift from the treatment, and birds foraged in the wheatfield to some degree. There appeared to be three waves of nesting attempts. The first observations were made of nests started at or near the time of spraying; the birds showed no territorial defense, were quiescent, and uttered no alarm cries. All nests were deserted. New nests with eggs were found about 2 weeks later and again after another week. One young was produced early, none later. Nests in control fields were much more successful. Eggs in the treated field contained 6 ppm of dieldrin,

and those in the control field contained 2 ppm. The authors emphasize the many problems associated with the study, including the fact that there was no opportunity for a pretreatment study, that disturbance affects nesting red-winged blackbirds, and that nest density in different hayfields normally varies. Thus the results must be judged as indicative rather than conclusive.

Wright (1965) reported that the breeding success of woodcock in DDT-treated areas of New Brunswick varied inversely with the amount of DDT used and with the area sprayed. With Nova Scotia used as a control, it appeared that the birds in the spray zone had a lower breeding success than those outside it.

Reproduction: Relation to Residues in Eggs and Adults

This section is intended as a summarization and discussion and hence includes extracts from some earlier sections in addition to new material.

Residue levels in eggs or tissues that indicate either hazard or safety have not been clearly defined, except in one study of trout. The relation between residues of DDT in trout eggs and mortality of fry was established by Burdick et al. (1964) through a combination of field and laboratory studies and experiments. The critical concentration was an amount in eggs equivalent to 2.9 ppm in fry, and no fry survived above 5 ppm.

It may or may not be possible to establish definitive relationships between residues in eggs and reproductive success of birds or reptiles. Hence, the indications from data now available are little more than suggestions for further research.

Among pheasants, minimal effects on reproduction have been associated with generally high residues in eggs and tissues. Variation in residue content has been high, so that correlations are difficult. As much as 263-473 ppm of DDT in egg yolks and 1,280 ppm in fat of adults was associated with a reduction of only 11 percent in chick survival (Azevedo et al., 1965). A 6-7 percent reduction prevailed when egg yolk residues were 13-133 ppm and fat residues were 40-460 ppm. In the field, egg yolk residues of DDT ranged from 129 to 1,181 ppm and fat residues from 1,542 to 3,647 ppm in areas where there were indications of some effects on reproduction (Hunt and Keith, 1962). Part of the adult population died in each of these studies. Chick survival was unaffected among pheasants whose eggs contained 46 and 52 ppm of dieldrin (Atkins and Linder, 1967). But in another study, pheasant chick survival was affected when eggs contained 3 and 193 ppm of dieldrin (Genelly and Rudd, 1956).

Herring gulls also apparently tolerate high residues of DDT in eggs and tissues (Keith, J. A., 1966; Hickey et al., 1966). Reproduction continued, although at a rate somewhat below normal in a Lake Michigan colony where eggs contained an average of 227 ppm (yolks approximately 363 ppm[3]) and adults averaged 2,441 ppm of DDT and its metabolites in the fat. Some DDT-induced mortality occurred among the adults.

Residue levels in successfully reproducing field populations provide some evidence of amounts that can be tolerated, but do not establish an upper limit. For example, eggs of successfully reproducing ospreys contained an average of 3 ppm of DDE (U.S. Bureau of Sport Fisheries and Wildlife, 1968). Arctic peregrines, in a population whose density had remained stable for 67 years and whose reproductive success was on the high side of the average, had about 27 ppm of DDT and metabolites and 1.3 ppm of dieldrin in muscle, and their eggs contained about 11 ppm of DDT and its metabolites and about 0.5 ppm of dieldrin[4] (Cade et al., 1968).

Cormorants in a stable, successfully reproducing colony in Muscongus Bay, Maine, had 6-11 ppm of DDE in composite egg samples (U.S. Bureau of Sport Fisheries and Wildlife, 1968; data of Channing Kury).

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[3] Computed, assuming yolks constituted approximately 16 percent of the weight of the egg contents.

[4] These are wet weight values converted from author's dry weight figures by dividing egg residues by 5 and muscle residues by 3.5.

Eggs of cormorants in inland lakes of mid-western United States and Canada in apparently successful colonies contained 11 ppm of organochlorine residues, primarily DDT and its metabolites (Anderson, 1967; U.S. Bureau of Sport Fisheries and Wildlife, 1967).

Disease

The effects of DDT on the immunological system were studied by Wassermann et al. (1967). Rats which received 200 ppm of p,p'-DDT in the drinking water for 35 days and 6 mg of ovalbumin in three injections showed increased liver weights, decreased spleen weights, and a 30 percent fall of antiovalbumin antibodies, in comparison with the control group. The sera of rats treated with p,p'-DDT showed a rise of albumin and a lowering of globulin fractions of serum proteins. The adrenals of animals subjected to surgical trauma (laparotomy), to unilateral gonadectomy or to hemithyroidectomy increased in weight. This phenomenon was almost completely inhibited in DDT-treated rats. These experiments suggest that the defense reactions are moderated in rats subjected to DDT ingestion.

Other relations between organochlorines and disease have been reviewed by Durham (1967). Production of cataracts in rats was related to long term feeding of heptachlor (Mestitzova, 1966). Carcinogenic potential of organochlorines has been the subject of various research studies and has been recently reviewed by Barnes (1966).

Nutrition

A relationship between toxicity and dietary variations was suggested by early studies of DDT. For example, Sauberlich and Baumann (1947) reported that rats and mice on a low fat and/or a high protein diet were more resistant to DDT than those on other diets. More recently, Phillips (1963) showed that the inclusion of 10 ppm of DDT in the diet of rats decreased the liver storage of vitamin A when carotene or vitamin A was orally administered. These effects were not produced by the inclusion of 0-20 ppm of dieldrin in the diet (Phillips, 1965). Lee et al. (1964) found that a protein-deficient diet accentuated the toxic effects of dieldrin.

The interaction of a toxic stress and a nutritional stress was studied in rats by Tinsley (1966). Diets were of three types: (1) nutritionally adequate, (2) with essentially no linoleic acid, and (3) with a marginal level of riboflavin. Rats received these diets with and without 20 ppm dieldrin. The addition of dieldrin to the diet lacking linoleic acid accentuated this fatty acid stress (as shown by the fatty acid composition of liver lipids), a result attributed to dieldrin interacting in the metabolism of polyunsaturated fatty acids. The riboflavin deficiency resulted in a depressed growth rate in female rats subjected to a dieldrin stress and appeared to accentuate dieldrin toxicity.

Long-term exposure of white-tailed deer to dieldrin at 25 ppm in the diet reduced growth rates of both fawns and yearlings, reduced reproductive rate of mature females, and reduced both birth weight and survival of fawns. Studies of characteristics suggested that the effects were mediated through nutritional abnormalities (Korschgen and Murphy, 1967). Chlordane resulted in growth abnormalities of rats (Ambrose et al., 1953).

Weight Loss and Other Stress

"Starvation depresses liver microsomal activity. Also, the protective effect afforded by the body fat, which serves to store the organochlorine insecticides and thus shield the sensitive nervous tissue, is either absent or is present in reduced amount. Thus, both these factors contribute to the increased susceptibility to pesticide poisoning which occurs in starved animals." (Durham, 1967).

Yet starvation may also result in enzyme stimulation when pesticides have been stored in the fat. In an experiment with rats, hepatic drug metabolism returned to normal levels 2 months after DDT feeding was discontinued, but even after that time hepatic microsomal enzymes were activated by starvation (Hart and Fouts, 1965).

Weight loss subsequent to dosage may produce delayed mortality among apparently healthy animals, as has been shown for several species, including sparrows (Bernard, 1963), rats (Dale et al., 1962; Barnes and Heath 1964; Heath and Vandekar, 1964), turkeys (Anderson et al., 1951), woodcock (Stickel, W. H., et al., 1965), and cowbirds (U.S. Bureau of Sport Fisheries and Wildlife, 1968).

Effect of change in length of day on mortality was shown in a group of Coturnix quail on a dietary dosage of 25 ppm of DDT for 6 months with no mortality. Molt and cessation of reproduction following day-length reduction resulting in death of 5 of 12 males and 3 of 11 females, but no deaths occurred among 24 controls (U.S. Bureau of Sport Fisheries and Wildlife, 1966; data of L. F. Stickel). In a similar comparison of Coturnix on a diet containing 10 ppm of dieldrin, two of three males and one of three females died after molting, and no deaths occurred among untreated birds (Stickel, W. H., et al., 1968).

In a study of cowbirds, the stress of disturbance was sufficient to bring on mortality. Birds on dosage of 40 ppm of DDT in the diet survived 8 weeks with loss of only 2 of 50 birds before a clean diet was restored. Additional mortality, including observed tremors, occurred 1, 8, 11, 15 (3 birds), and 28 days postdosage, and six of these seven deaths followed the disturbance of entering the cage or the one adjacent for the purpose of catching birds (Stickel, W. H., 1965).

Behavior

Changes in behavior or nervous system function have been produced by certain organochlorines. Endrin injected into the blood stream of pigeons increased certain brain-wave amplitudes at a dosage about 30 percent of the ED 50 for seizure activity (Revzin, 1966). The areas affected were those related to vision. Dési et al. (1966) showed that DDT fed to rats with the diet produced changes in frequency and amplitude of brain waves. The changes at the higher dose (40 mg/kg/da) appeared on the 6th day, the same time as ataxic symptoms; those at the lower dose (20 mg/kg/da) appeared 3 weeks earlier than ataxic symptoms. Rats on the lower doses showed no changes in maze-running ability. Khäiry (1959, 1960) similarly found that sublethal dietary doses of DDT and dieldrin did not affect running time and problem solving by rats.

These results suggest the need for thorough and exhaustive studies of effects of organochlorines on nervous system responses of wild species, and their possible effects under natural conditions. Responses that could have profound effects have been reported for fish. New Brunswick salmon from DDT-sprayed rivers proved to be unusually sensitive to low temperatures, and very low doses resulted in shifts in temperature selection of sufficient magnitude to have serious implications for survival (Ogilvie and Anderson, 1965). A temperature relationship to DDT effects also was shown for rats: amounts that did not otherwise produce symptoms reduced the ability of the rats to swim in cold water (20° C) (Durham, 1967). Hypersensitivity of bluntnose minnows and guppies as a result of endrin exposure is discussed by Mount (1962). Goldfish exposed to toxaphene in the water at about 16 percent of the 96-hour TLm were more active than controls and learned more rapidly (Warner et al., 1966). The practical importance of hyper-irritability is ambiguous. If a fish in nature fails to hide itself effectively because of over-response to the close quarters of a shelter, it may be eaten, but if it notices its enemy sooner than most, it may escape. Changes, however, provide a warning and a clue even if they cannot properly be interpreted as good or bad.

Decline of Scottish golden eagles in recent years was associated with increased egg breakage, and the hypothesis was proposed that this pathological behavior resulted from organochlorine contamination. Sparrowhawks and peregrines also destroyed their eggs in areas where there was a marked population decline (Lockie and Ratcliffe, 1964; Ratcliffe, 1963).

Nesting herring gulls in Lake Michigan that were heavily contaminated with organochlorines produced eggs with defective shells, and

the investigator also suspected a relation between restlessness of adults and egg damage (Keith, 1966a). On another Lake Michigan island where gulls contained high DDT residues, there was a higher incidence of egg breakage and the gulls showed a greater degree of aggressiveness than did those on an island where the birds contained lower residues (Ludwig and Tomoff, 1966).

The reduction of egg shell thickness reported by Ratcliffe (1967) could well enhance egg breakage, whether accidental or the result of altered behavior of the adults.

DISCUSSION

The Problem of Appraisal

Organochlorine pesticides have become integral components of the biological system. Their manufacture and use are widespread and continuing. These facts have stimulated a very large effort to measure the amounts and kinds of organochlorines now in existence in man and his environment, including the other animals, domestic and wild, that share his occupancy of the earth. The avowed purpose of this enormous factgathering effort is to provide a baseline for determining the trends of residues with time. Obviously, this inquiry is not an academic one. Results are expected to serve as an alert to hazard. Unfortunately, the significance of these residues is not well understood.

Hence, an even more enormous effort is being made to understand and interpret the effects of these pesticides. The greatest part of this effort is in physiological, pharmacological, and toxicological studies whose results are most clearly important to understanding direct effects on the health of man. Other effects on man's world have received less scientific scrutiny. Results of some studies on the effects of organochlorine pesticides on the natural world have been reviewed in this paper. A full appraisal is not yet possible.

A Critical Question

Present knowledge demonstrates the existence of pesticide residues nearly everywhere. Transport through the ecological system and biological accumulation in animals are evident wherever sufficient examination is made. Degradation and loss of organochlorines from the evironment proceeds slowly. The impact of these new components of the ecosystem appears as death, reproductive impairment, disruption of species balance, and behavioral alteration, but the overall effects on the environment have not been determined.

Early failures to diagnose pesticide-induced mortality from residue content were overcome by adoption of experimental procedures, Brain residues indicative of hazard of death from DDT and dieldrin poisoning were determined. Studies of relations between residue content and physiological effects such as enzyme induction, shell thinning, and other factors that may affect reproduction have only begun.

Research Needs

Three types of research effort appear paramount:

1. Interpretation of significance. Research is needed to determine the quantities of pesticides in animals and their environments that indicate potentially deleterious effects. These interpretations require experimental studies especially designed for the purpose. Diagnostic tissues for identification of lethal and sublethal effects should be determined and toxic limits established for the various chlorinated hydrocarbons and heavy metals together and separately. Variations due to physiological and environmental stresses should

be established. Species differences in sensitivity should be determined. Research to determine residue levels indicative of hazard to normal reproduction and behavior should receive special emphasis.

2. Environmental assessment. Quantities of residues in the environment should be determined. General environmental quantities and trends determined in the National Monitoring Program provide a part of this information. More detailed studies are required of environments of declining species such as eagles, pelicans, and ospreys, and of truly rare and endangered forms. Parallel studies are required in important wildlife habitats. Estuarine areas, inland waters, agricultural lands, forests, and grazing lands all historically have supported significant wildlife populations. Investigations should be perceptive, rather than general; they should focus on the specific habitats and on the food organisms actually consumed in these habitats.

3. Environmental kinetics. Rates and routes of gain, loss, and change of residues in and among the various living and nonliving components of the environment are required to identify sites of hazard or to gain assurance of lack of hazard.

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